CASE REPORT / OLGU SUNUMU

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Posterior Reversible Encephalopathy Syndrome (PRES) in a Patient Who Developed Visual Loss After Total Thyroidectomy: A Case Report

Total Tiroidektomi Sonrası Görme Kaybı Gelişen Hastada Posterior Reversible Ensefalopati Sendromu (PRES): Bir Olgu Sunumu

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Abstract

Posterior reversible encephalopathy syndrome (PRES) is a rare but potentially severe neurological disorder most commonly associated with pregnancy toxemia (preeclampsia and eclampsia), hypertension, acute glomerulonephritis, and cytotoxic or immunosuppressive agents. Clinical manifestations typically include headache, visual disturbances, altered mental status, and generalized or focal seizures, often accompanied by hypertension. Diagnosis is based on a detailed neurological examination and neuroradiological investigations, particularly cranial magnetic resonance imaging. In this case report, we present PRES and describe its clinical presentation in a 40-year-old male patient with no known comorbidities; the condition developed secondary to head hyperextension following total thyroidectomy.

Keywords: Thyroidectomy, visual loss, posterior reversible encephalopathy syndrome

Öz

Posterior reversible ensefalopati sendromu (PRES); çoğunlukla gebelik toksemisi (preeklampsi/eklampsi), hipertansiyon, akut glomerülonefrit, sitotoksik ve immünosüpresif ajan kullanımı ile ilişkili olarak gelişen, nadir ancak potansiyel olarak ciddi seyir gösterebilen bir nörolojik sendromdur. Klinik tabloda sıklıkla baş ağrısı, görme bozuklukları, mental durum değişiklikleri ve jeneralize veya fokal nöbetler izlenmekte olup bu bulgulara çoğu zaman eşlik eden hipertansiyon bulunmaktadır. Tanı; ayrıntılı nörolojik muayene ile birlikte nöroradyolojik görüntüleme yöntemleri, özellikle kraniyal manyetik rezonans görüntüleme bulguları temel alınarak konulmaktadır. Bu olgu sunumunda, total tiroidektomi sonrası başın hiperekstansiyonu sonucu gelişen ve bilinen herhangi bir ek hastalığı bulunmayan 40 yaşında bir erkek hastadaki press sendromunu ve klinik belirtilerini sunuyoruz.

Anahtar Kelimeler: Tiroidektomi, görme kaybı, posterior reversible ensefalopati sendromu

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Introduction

Posterior reversible encephalopathy syndrome (PRES) is a rare but potentially serious neurological disorder that affects the central nervous system. Although it is more commonly observed in the adult population, it has also been described in pediatric cases (1,2). The most common etiological factors include pregnancy toxemia (preeclampsia/eclampsia), acute or uncontrolled hypertension, acute glomerulonephritis, and the use of cytotoxic or immunosuppressive agents; sepsis and autoimmune diseases are also considered predisposing factors. The clinical presentation is characterized by headache, visual disturbances, altered mental status, and generalized or focal seizures, often accompanied by hypertension (3).

In the pathophysiology of PRES, hyperperfusion resulting from impaired cerebral autoregulation and/or endothelial dysfunction leads to disruption of the blood-brain barrier, increased capillary leakage, and ultimately vasogenic cerebral edema (4). Diagnosis is based on a combination of detailed medical history, clinical findings, and neuroradiological imaging. Computed tomography (CT), and cranial magnetic resonance imaging (MRI) in particular, typically reveal bilateral, symmetric areas of edema, most commonly involving the occipital and parietal lobes, which are characteristic of the diagnosis.

When the condition is diagnosed early and the underlying cause is promptly eliminated with appropriate treatment, patients often show a complete and dramatic recovery. However, delayed diagnosis and treatment may result in serious complications such as cerebral ischemia, infarction, permanent neurological deficits, and even death (2).

In this report, we aim to present a patient who developed PRES in the early postoperative period following total thyroidectomy for thyroid cancer and who was discharged without sequelae after early diagnosis and appropriate treatment, in light of the existing literature.

Case Presentation

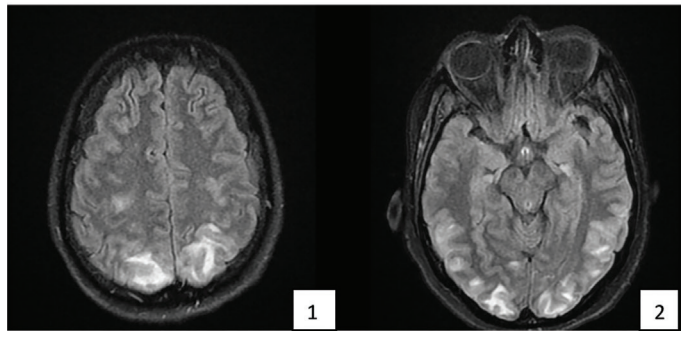
A 40-year-old male patient presented to our clinic after a thyroid nodule was incidentally detected on CT performed to evaluate a cough. Thyroid function tests were within normal limits. Neck ultrasonography revealed a 1-cm TIRADS 5 nodule in the left thyroid lobe and an 8-mm TIRADS 3 nodule in the right thyroid lobe. Fine-needle aspiration biopsy of the left lobe was consistent with papillary thyroid carcinoma. The patient was prepared for surgery, and the vocal cords were examined. At the patient's request, total thyroidectomy was performed. The patient gave informed consent for total thyroidectomy.

No intraoperative complications occurred. Postoperatively, serum calcium, parathyroid hormone, and electrolyte levels were within normal limits. The patient's postoperative course was uneventful until the 4th postoperative hour, when presyncope and sudden-onset bilateral visual loss were noted. The patient was transferred to the intensive care unit. The initial blood pressure measurement was 170/100 mmHg. Arterial blood gas analysis revealed no significant abnormalities, and detailed hematological and biochemical tests were unremarkable.

Neurological examination showed no lateralizing motor or sensory deficits. Deep tendon reflexes were globally brisk (+++). The patient reported a complete loss of vision. On visual acuity examination, there was no blink response to threat or to finger counting, and no light perception was detected in either eye.

A non-contrast cranial CT scan performed to rule out hemorrhage revealed no acute bleeding. The patient was referred to the neurology department. Cranial MRI and contrast-enhanced brain and neck CT angiography were requested. CT angiography demonstrated normal carotid arteries and branches without thrombosis or stenosis. MRI revealed cortical and subcortical hyperintense areas on T2-weighted and FLAIR sequences in the bilateral parieto-occipital lobes (Figures 1-2). These findings were reported as consistent with PRES.

Based on clinical findings and imaging studies, a diagnosis of PRES secondary to head hyperextension during surgery was established. The patient was started on amlodipine 10 mg and dexamethasone 8 mg, the latter administered four times daily. Laboratory values remained stable during follow-up. The patient was transferred to the ward on the second day. On the fifth day, neurological reevaluation revealed improvement on visual examination, with the return of the blink response to threat and bilateral finger counting. It was recommended that steroid therapy be tapered and discontinued while antihypertensive treatment was continued. The patient was discharged in good condition on postoperative day 7, in accordance with the neurology recommendations.



Figures 1-2. Brain MRI images of the patient showing hyperintense areas in the parieto-occipital regions

MRI: Magnetic resonance imaging

Discussion

PRES is a rare but potentially serious neurological disorder presenting with headache, altered mental status, visual loss, and generalized or focal seizures. Although its pathophysiology has not been fully elucidated, it is most commonly associated with bilateral cerebral edema predominantly involving the occipital and parietal regions (5). While the clinical course is often transient and reversible, severe morbidity and mortality may occur due to complications. The syndrome was first described in 1996 by Hinchey et al. (4). The most common etiological factors include hypertension, eclampsia, and the use of immunosuppressive or cytotoxic drugs. Additionally, collagen vascular diseases, human immunodeficiency virus infection, celiac disease, chronic renal failure, sepsis, and organ transplantation have also been reported as contributing factors (4).

An initial hypothesis of the pathophysiology of PRES proposed that acute, severe hypertension causes cerebral vasoconstriction, leading to hypoperfusion and cytotoxic edema. However, the more widely accepted theory today proposes that hypertension-induced disruption of cerebral autoregulation results in hyperperfusion, blood-brain barrier breakdown, and subsequent vasogenic edema (6). In the present case, positional hypoxia resulting from head hyperextension during thyroidectomy, followed by reperfusion injury, was considered a distinctive and noteworthy etiological mechanism leading to vasogenic edema and PRES.

Diagnosis of PRES relies on high clinical suspicion following thorough history-taking and physical examination, with neuroradiological imaging playing a critical role in confirmation. CT, and especially cranial MRI, typically demonstrate edema predominantly affecting the posterior cerebral hemispheres, particularly the occipital and parietal regions, appearing as hyperintense lesions on T2-weighted and FLAIR sequences. The presence of bilateral cortical and subcortical hyperintense lesions on MRI is highly valuable for diagnosis (7-9). Differential diagnoses include posterior circulation infarctions, encephalitis, cerebral edema secondary to hyponatremia, demyelinating diseases, and cerebral venous thrombosis.

Early diagnosis and prompt treatment are the most important determinants of prognosis in PRES. Delayed management may result in irreversible brain damage, cerebral ischemia, chronic epilepsy, or death (1). Treatment primarily aims at controlled blood pressure regulation, effective seizure management, reduction of cerebral edema, and elimination of the underlying etiological factor (1,10). Calcium channel blockers and organic nitrates are commonly used for antihypertensive therapy; however, rapid and excessive blood pressure reduction should be avoided due to the risk of cerebral infarction and organ dysfunction.

Because cerebral edema is the central pathological feature, anti-edema therapy is essential. Benzodiazepines and phenytoin are frequently used to control generalized seizures, while refractory cases may require intubation and intensive care support. In cases of PRES associated with preeclampsia, magnesium sulfate is preferred as an anticonvulsant following blood pressure control, as it does not cross the blood-brain barrier and is considered safe during pregnancy (11). In patients with persistent or worsening symptoms, cesarean section may become unavoidable to improve maternal and fetal outcomes.

A review of the literature shows that publications and case reports on PRES indicate that the syndrome is predominantly observed in pregnant women with preeclampsia/eclampsia and in patients with autoimmune connective tissue diseases. Only a single case report has been published following thyroidectomy, in which the development of press syndrome was attributed to excessive calcium replacement administered for the treatment of postoperative hypoparathyroidism (12). In contrast, the primary cause of press syndrome in our case was ischemia-reperfusion injury secondary to head hyperextension. From this perspective, our case report provides a novel contribution to the existing literature.

Conclusion

Although rare, PRES can cause serious neurological morbidity and mortality if not diagnosed and treated promptly. Its clinical presentation typically includes headache, altered consciousness, visual disturbances, and seizures, which collectively may mimic many other neurological disorders; therefore, high clinical awareness is essential, particularly in patients with risk factors.

Diagnosis is primarily based on clinical suspicion supported by neuroradiological imaging, especially cranial MRI findings of bilateral cortical and subcortical vasogenic edema in the posterior regions. Early imaging plays a crucial role in preserving the syndrome's reversibility and preventing complications.

In the present case, positional hypoxia caused by intraoperative head hyperextension followed by reperfusion injury-an infrequently reported mechanism-was considered a noteworthy etiological factor for PRES. This highlights that even in the absence of classical risk factors such as hypertension, perioperative cerebral perfusion disturbances may predispose patients to PRES. Therefore, appropriate patient positioning and maintenance of cerebral oxygenation during surgical procedures are essential preventive measures.

Treatment focuses on blood pressure control, seizure control, reduction of cerebral edema, and elimination of the underlying cause. With this comprehensive approach, most patients achieve complete clinical and radiological recovery. However, delayed diagnosis and treatment may result in irreversible brain injury, chronic epilepsy, or death.

Early recognition of PRES, inclusion in the differential diagnosis, and prompt implementation of etiologically targeted treatment strategies are the most critical factors determining patient prognosis. Reporting atypical etiological mechanisms contributes to a better understanding of the syndrome's pathophysiology and increases clinical awareness.

Ethics

Informed Consent: The patient gave informed consent for total thyroidectomy.

Footnotes

Authorship Contributions

Concept/Design: K.K.Ö., İ.B.B., M.A.G., Data Collection or Processing: K.K.Ö., M.T., Analysis or Interpretation: İ.B.B., M.A.G., Literature Review: M.T., Writing, Reviewing and Editing: K.K.Ö.

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